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S1	15	"5166137"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:27
S2	2	Bjerkvig-r\$.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:30
S3	52659	alginate	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:34
S4	23521	S3 and cell	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:34
S5	9776	S4 and tumor	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:34
S6	6815	S5 and (CNS brain)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:35
S7	2677	S5 and ((CNS brain) SAME tumor)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:35
S8	1847	alginate WITH cell	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:35
S9	169	S8 AND ((CNS Brain) WITH tumor)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:35
S10	30	S9 and (endostatin angiostatin thrombospondin prolactin)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:36
S11	1	S10 and microbead	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/21 13:36



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☐ 1: Ann N Y Acad Sci. 1999;886:58-66.

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Antiangiogenic domains shared by thrombospondins and metallospndins, a new family of angiogenic inhibitors.

Iruela-Arispe ML, Vazquez F, Ortega MA.

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The growth of solid tumors has been shown to depend on neovascularization. By understanding the mechanisms that control the neovascular response, it may be possible to design therapeutic strategies to selectively prevent or halt pathologic vascular growth and restrain cancer progression. Thrombospondin-1 is an extracellular matrix protein that among several functions suppresses capillary growth in angiogenesis assays. We have demonstrated that within the context of the mammary gland TSP1 can modulate normal development of blood vessels. Expression of TSP1 in transgenic animals under the control of the MMTV promoter was associated with a 50-72% reduction in capillary growth. In addition, TSP1 reduced tumor size in transgenic overexpressors. The data suggest an important role for TSP1 in modulating vascular growth in both normal and pathologic tissues. The antiangiogenic region of TSP1 has been mapped to the type I (properdin) repeats. To identify novel proteins with such a domain, we have cloned two cDNAs (METH-1 and METH-2) which also have antiangiogenic properties. In addition to carboxyterminal thrombospondin-like domains they also contain metalloproteinase and disintegrin sequences. Expression of both proteins is broad but nonoverlapping. Recombinant fragments from these sequences have strong antiangiogenic potential in the CAM and cornea pocket assays. At the same molar ratio, METH-1 and METH-2 are about 20-fold more potent than TSP1. We predict that these proteins are likely endogenous modulators of vascular growth with relevant therapeutic potential in cancer and other disease states.

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1: Endocrinology. 1993 Sep;133(3):1292-9.

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endo.endojournals.org**The 16-kilodalton N-terminal fragment of human prolactin is a potent inhibitor of angiogenesis.**

Clapp C, Martial JA, Guzman RC, Rentier-Delure F, Weiner RI.

Reproductive Endocrinology Center, University of California, San Francisco 94143.

The formation of a new blood supply, angiogenesis, is an essential component of carcinogenesis and unrestricted tumor growth. A substance capable of inhibiting angiogenesis would be of considerable therapeutic potential in the treatment of cancer. We previously reported that the 16-kilodalton N-terminal fragment of rat PRL (16K rPRL) was a potent inhibitor of capillary endothelial cell proliferation via a novel receptor. We now report that the nanomolar concentrations of recombinant human 16K PRL inhibit basal and basic fibroblast growth factor or vascular endothelial growth factor-stimulated growth of bovine brain capillary endothelial cells. 16K human (h) PRL also inhibits stimulation of human umbilical vein endothelial cell proliferation by basic fibroblast growth factor. The organization of endothelial cells into capillary-like structures in type I collagen gels is also prevented by 16K hPRL. Furthermore, in an in vivo assay, the chick embryo chorioallantoic membrane assay, 16K hPRL as well as 16K rPRL were potent inhibitors of capillary formation. 16K hPRL, like 16K rPRL, maintains its biological activity as a partial PRL agonist at PRL receptors on mammary gland epithelial cells. These data demonstrate for the first time that the biological activity of 16K rPRL is not unique and that similar fragments of hPRL are active. The antiangiogenic activity of these molecules is conserved across avian and mammalian species. That 16K hPRL is a potent antiangiogenic factor in in vitro and an in vivo assay raises the exciting potential of this peptide being capable of inhibiting tumor growth.

PMID: 7689950 [PubMed - indexed for MEDLINE]

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Endostatin: a promising drug for antiangiogenic therapy.

Cirri L, Donnini S, Morbidelli L, Chiarugi P, Ziche M, Ledda F.

Department of Pharmacology, University of Firenze, Italy.

Angiogenesis, the formation of new blood vessels from existing capillaries, is critical for tumors to grow beyond a few in size. Tumor cells produce one or more angiogenic factors including fibroblast growth factor and vascular endothelial growth factor. Surprisingly, antiangiogenic factors or angiogenesis inhibitors have been isolated from tumors. Some angiogenesis inhibitors, such as angiostatin, are associated with tumors while others, such as platelet-factor 4 and interferon-alpha are not. Endostatin, a C-terminal product of collagen XVIII, is a specific inhibitor of endothelial cell proliferation, migration and angiogenesis. The mechanism by which endostatin inhibits endothelial cell proliferation and migration is unknown. Endostatin was originally expressed in a prokaryotic system and, later, in a yeast system, thanks to which it is possible to obtain a sufficient quantity of the protein in a soluble and refolded form to be used in preclinical and clinical trials.

Publication Types:

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